Focus sur le potassium

Physiologie rénale

Intégration de l'homéostasie hydro-électrolytique par les hormones

- Valeurs normales d'apport de sodium, distribution du sodium dans l'organisme, voies d'excrétion du sodium
- Valeurs normales d'apport de potassium, distribution du potassium dans l'organisme, voies d'excrétion du potassium
- Mécanisme d'action et effets de l'aldostérone, et du système rénine angiotensine
- Contrôle hormonal des pools sodique et potassique

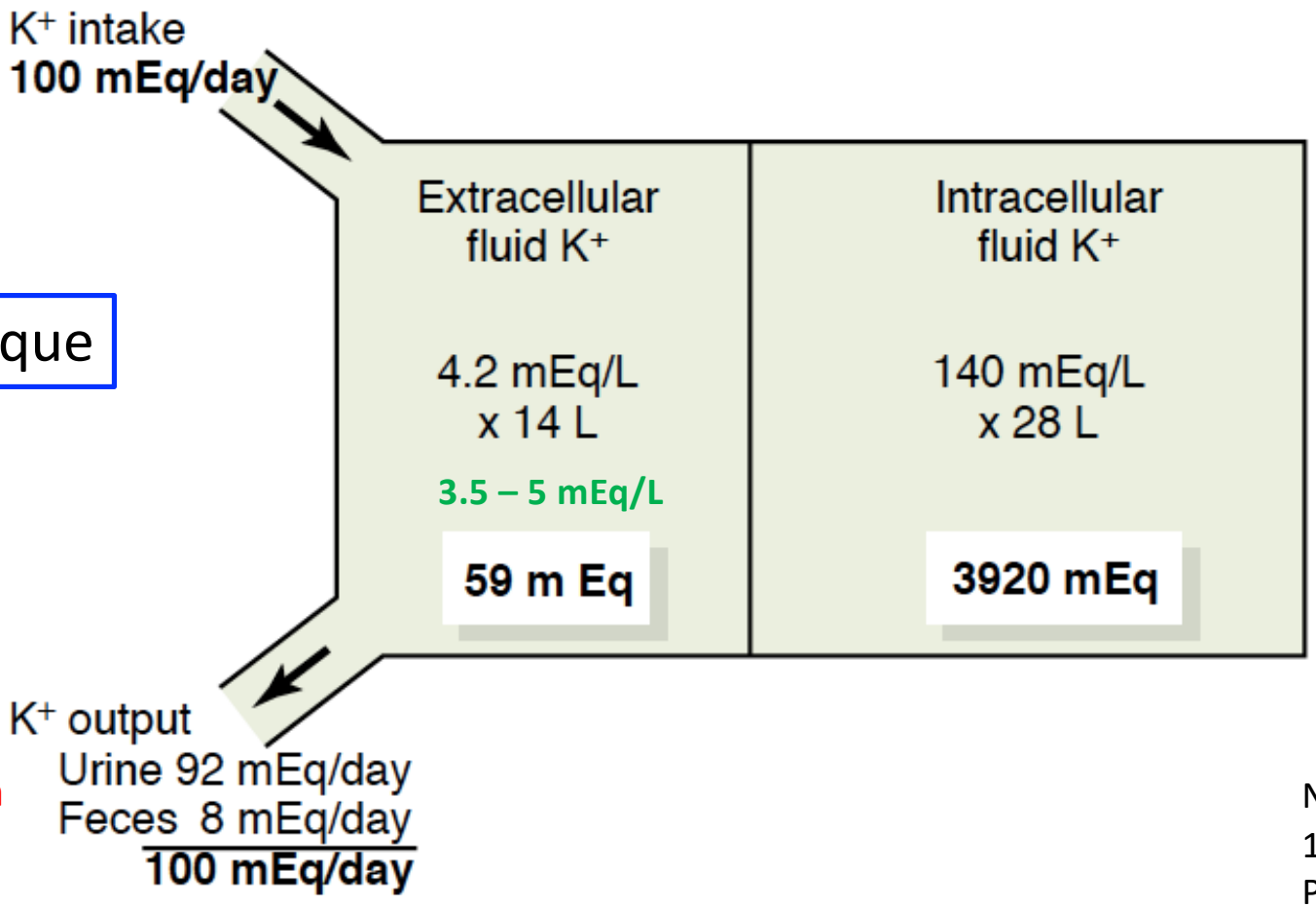
rénale >90%
fécale < 10%

Facteurs de régulation de la sécrétion de l'aldostérone, de la rénine, de l'angiotensine

Cellule de la zone glomérulée

- kaliémie
- angiotensine II
- ANP (Atrial Natriuretic Peptide)
- ACTH

Bilan potassique



Voies d'excrétion

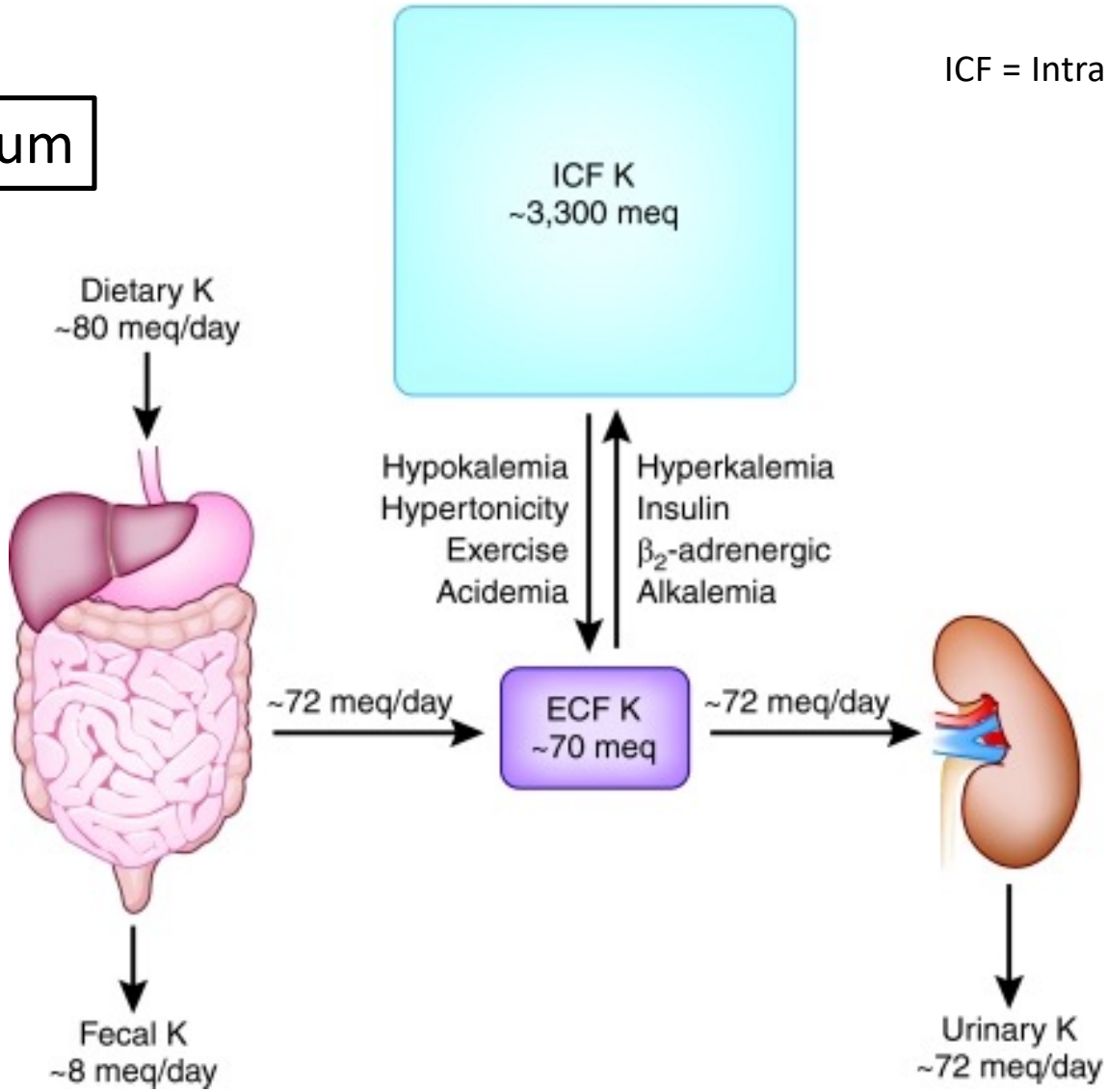
Note :
14 L + 28 L = 42 L
Personne pesant 60 kg
70% de 60 kg = 42 L

Figure 29–1

Normal potassium intake, distribution of potassium in the body fluids, and potassium output from the body.

Bilan Potassium

ICF = Intra Cellular Fluid



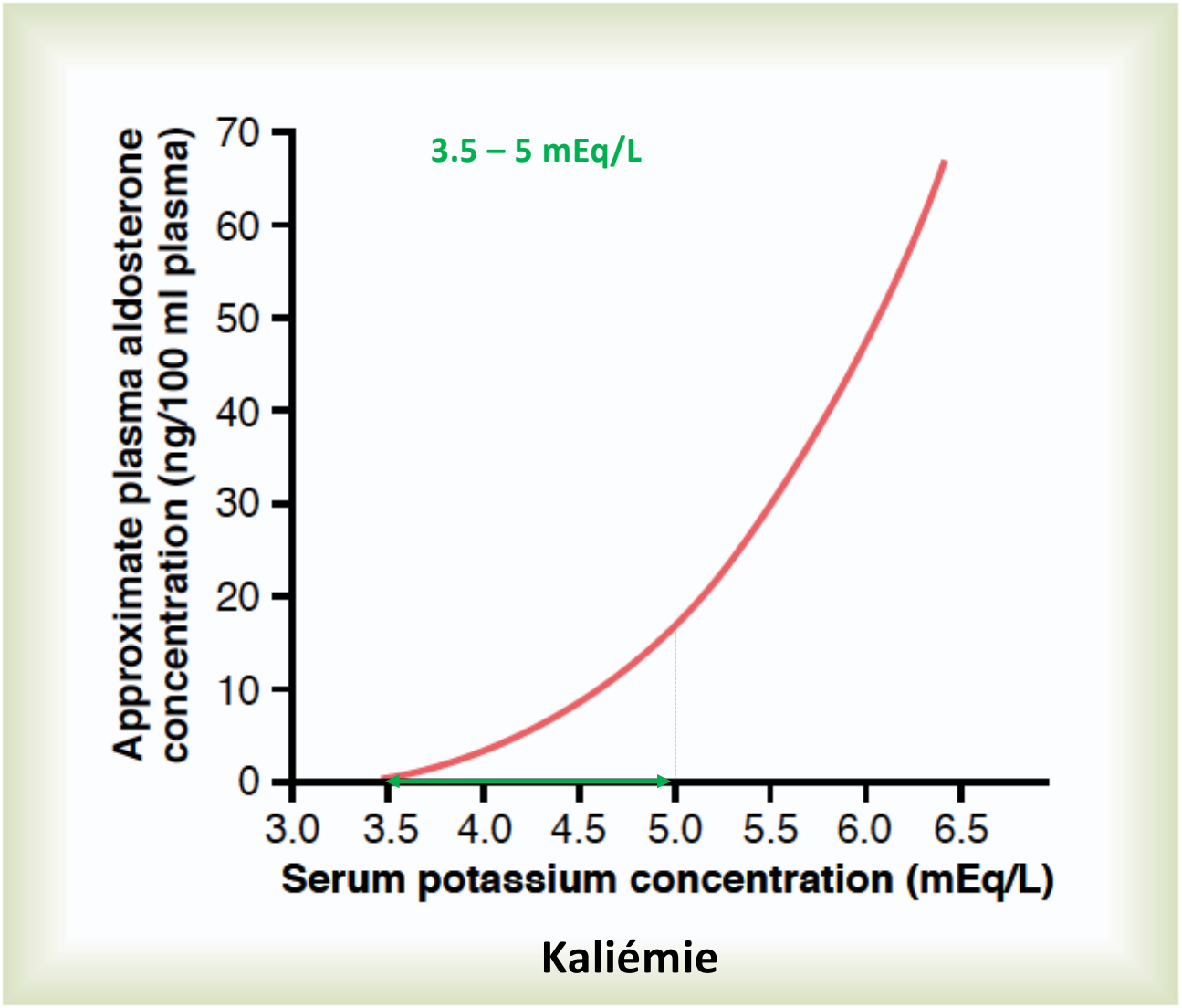


Figure 29–5

Effect of extracellular fluid potassium ion concentration
on plasma aldosterone concentration.

Note that small changes in potassium concentration cause large changes in aldosterone concentration.

4.25 ± 0.75 mmol /l (mEq/L)

Homéostasie de la kaliémie

Effect of large changes in **potassium intake** on extracellular fluid potassium concentration under normal conditions (*red line*) and after the aldosterone feedback had been blocked (*blue line*).

Note that after blockade of the aldosterone system, regulation of potassium concentration was greatly impaired.
(Courtesy Dr. David B. Young.)

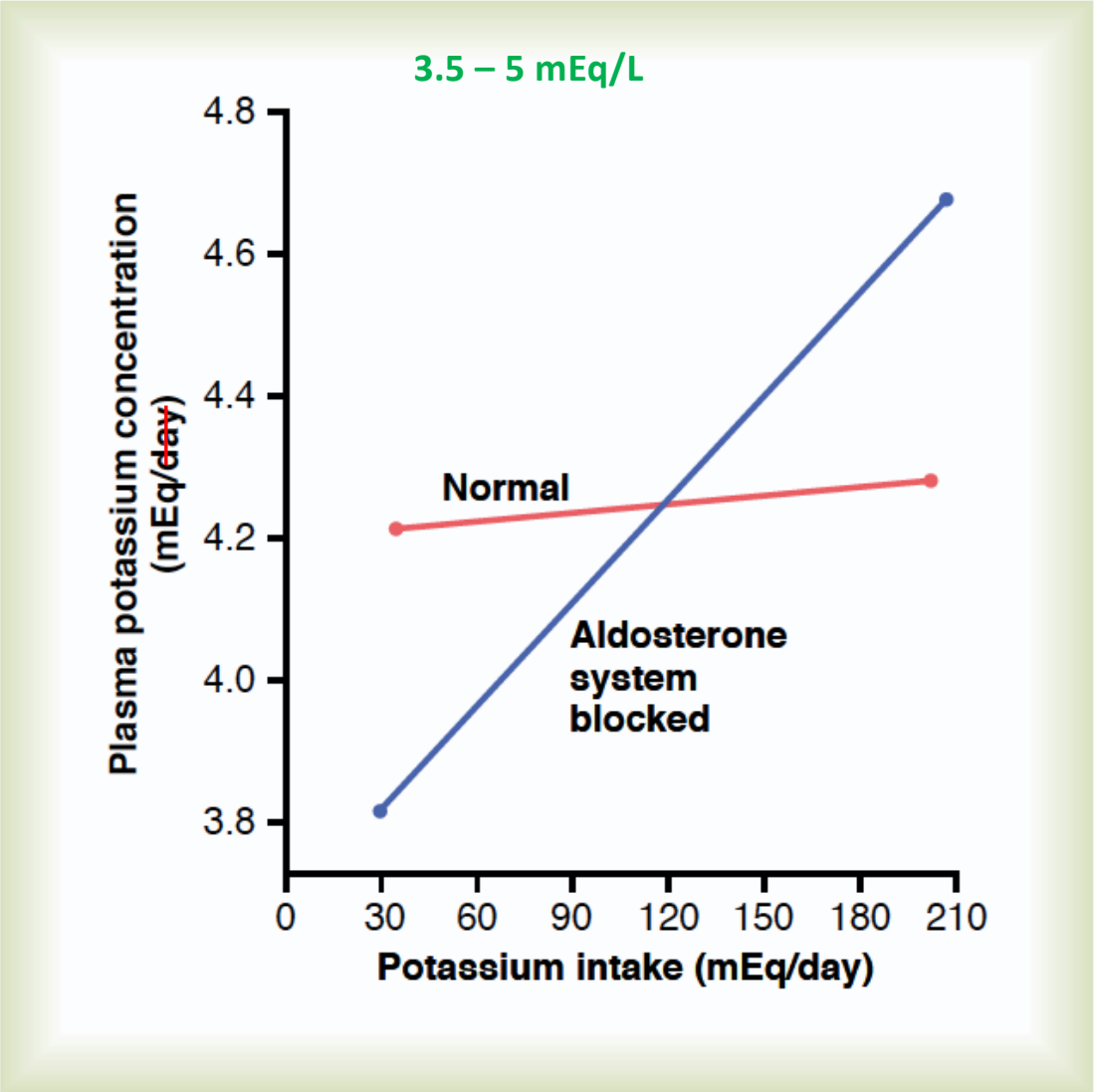


Figure 29–8 chien

L'aldostérone stimule la sécrétion rénale de K^+

Effect of
plasma aldosterone concentration (*red line*)
and
extracellular potassium ion concentration (*black line*)
on the rate of urinary potassium excretion.

These factors stimulate potassium secretion
by **the principal cells** of the cortical collecting tubules.

(Drawn from data in Young DB, Paulsen AW:
Interrelated effects of aldosterone and plasma
potassium on potassium excretion.
Am J Physiol 244:F28, 1983.)

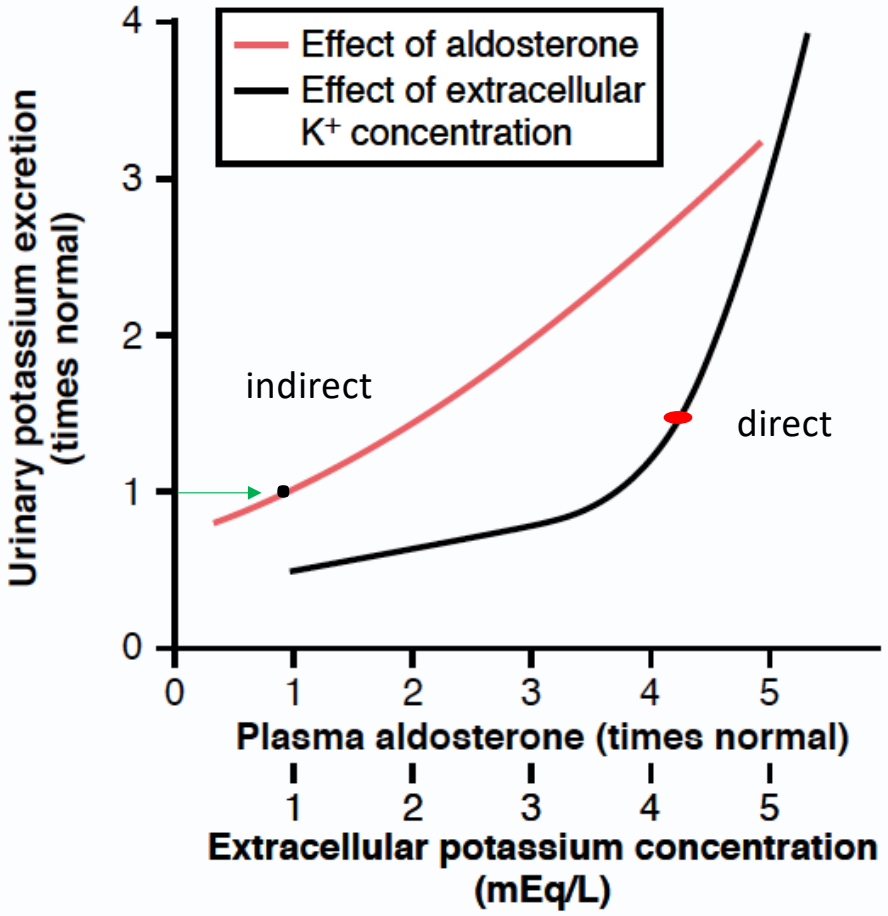


Figure 29–4

Homéostasie de la kaliémie

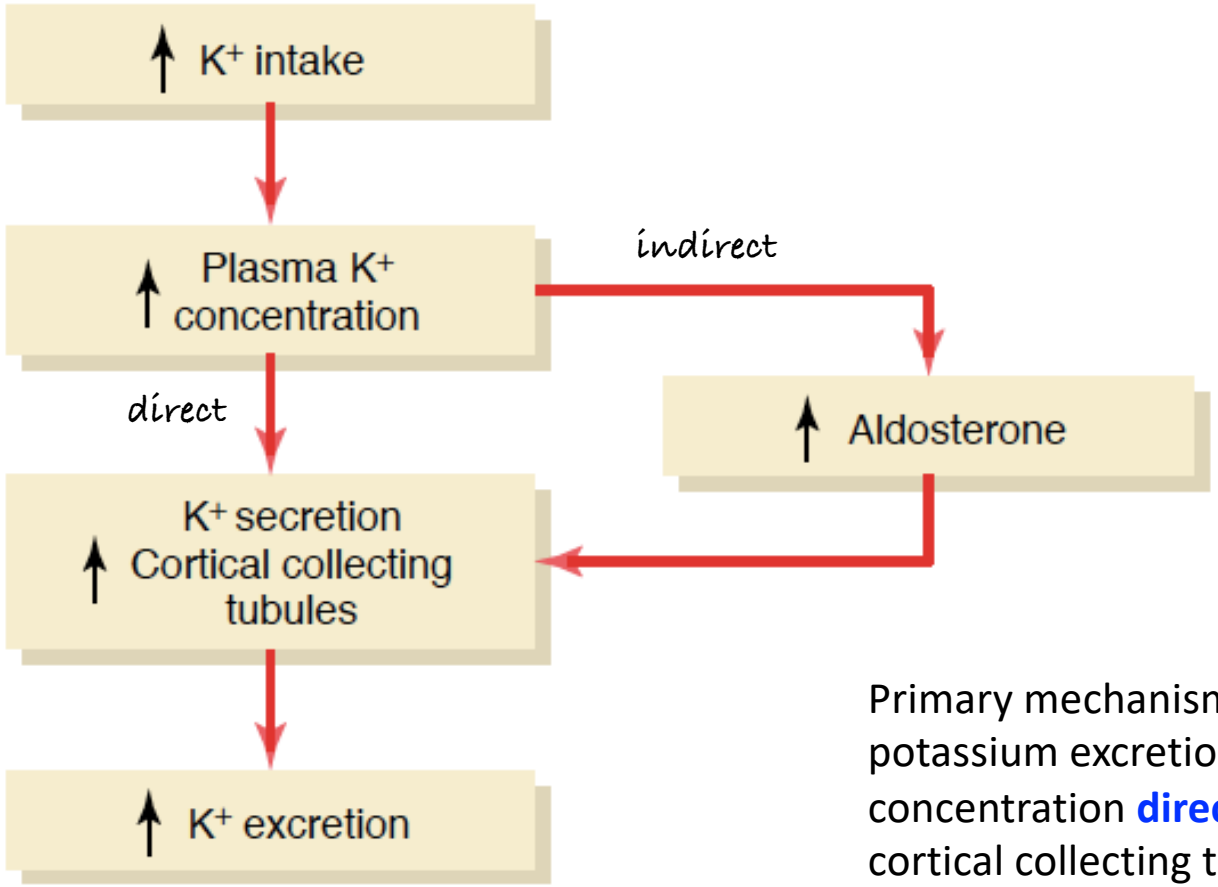
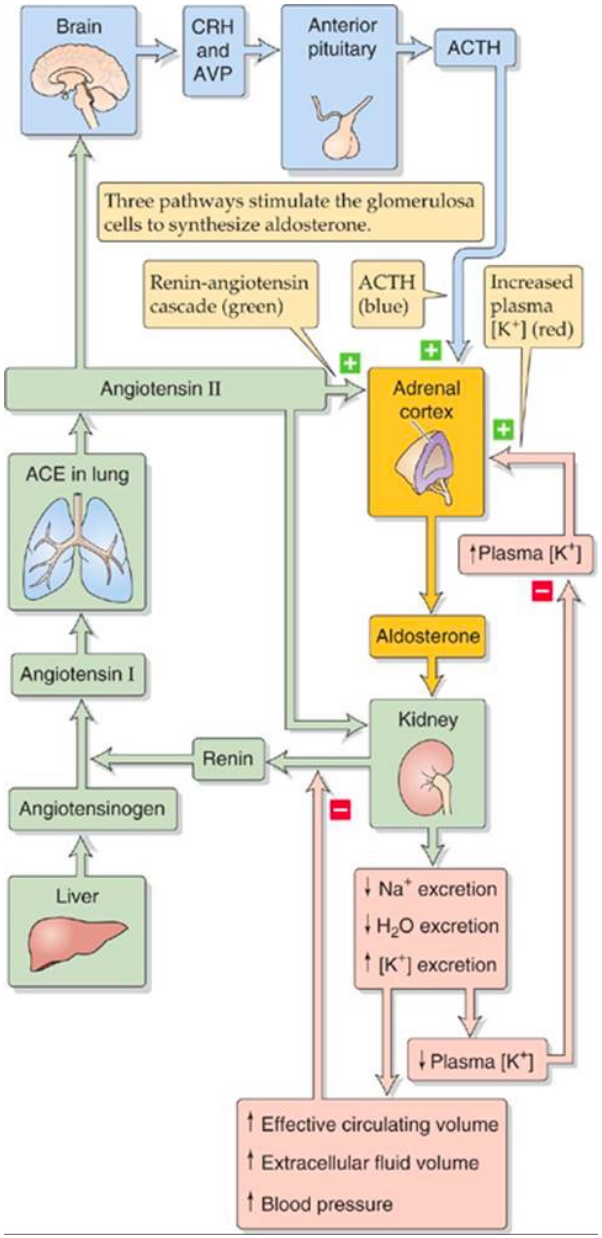
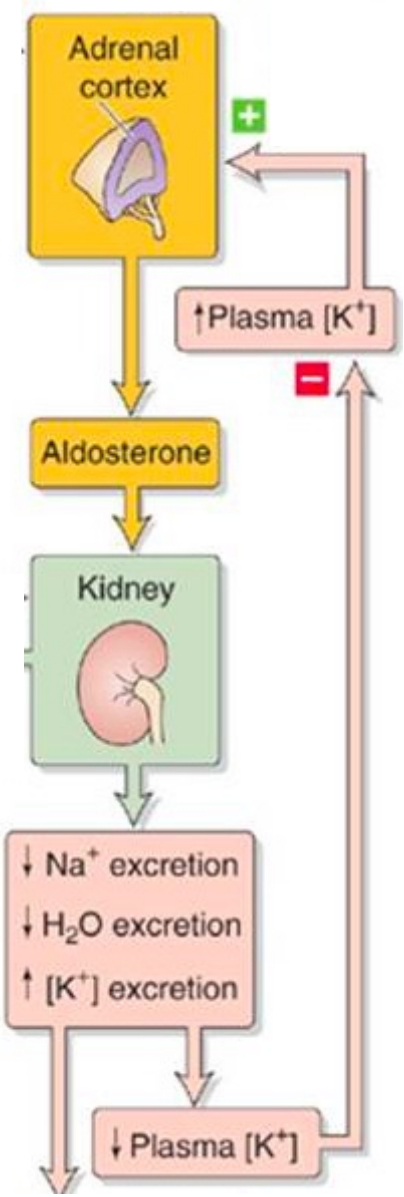


Figure 29–7

Primary mechanisms by which high potassium intake raises potassium excretion. Note that increased plasma potassium concentration **directly** raises potassium secretion by the cortical collecting tubules and **indirectly** increases potassium secretion by raising plasma aldosterone concentration.



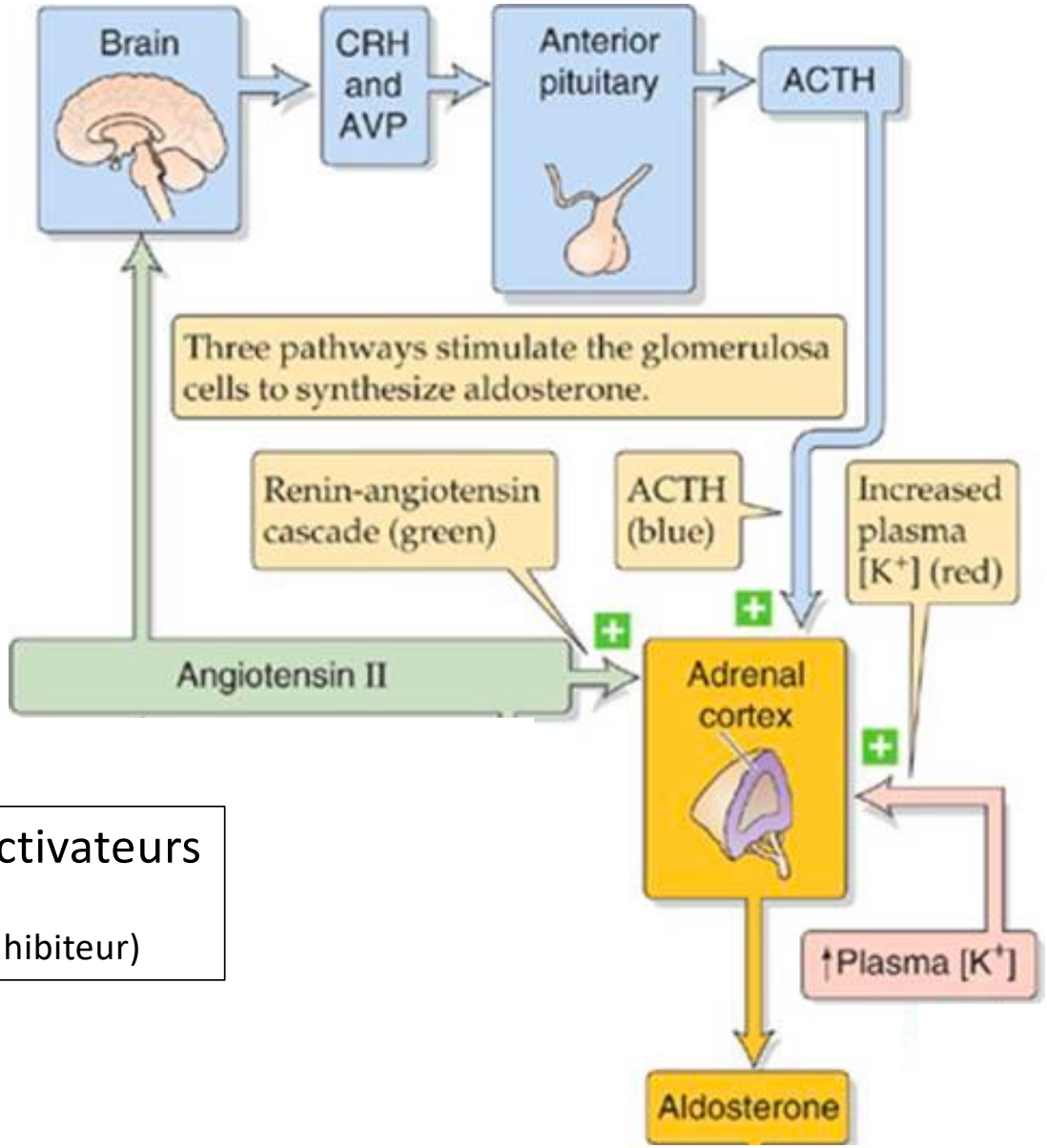


Boucle de régulation de la Kaliémie

3.5 – 5 m Eq/L

Boron

L'importance de la voie indirecte est douteuse.



3 stimuli activateurs

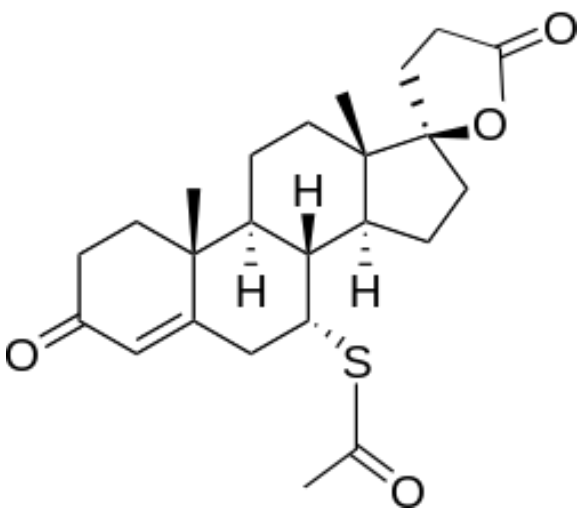
(ANP est un inhibiteur)

kaliémie **3.5 – 5 m Eq/L**

Boron

Antagonistes de l'aldostérone

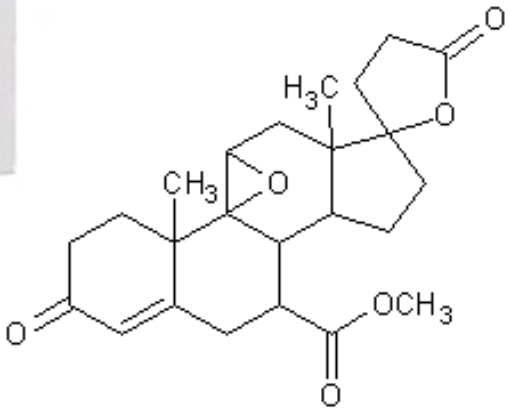
lactone



stéroïde

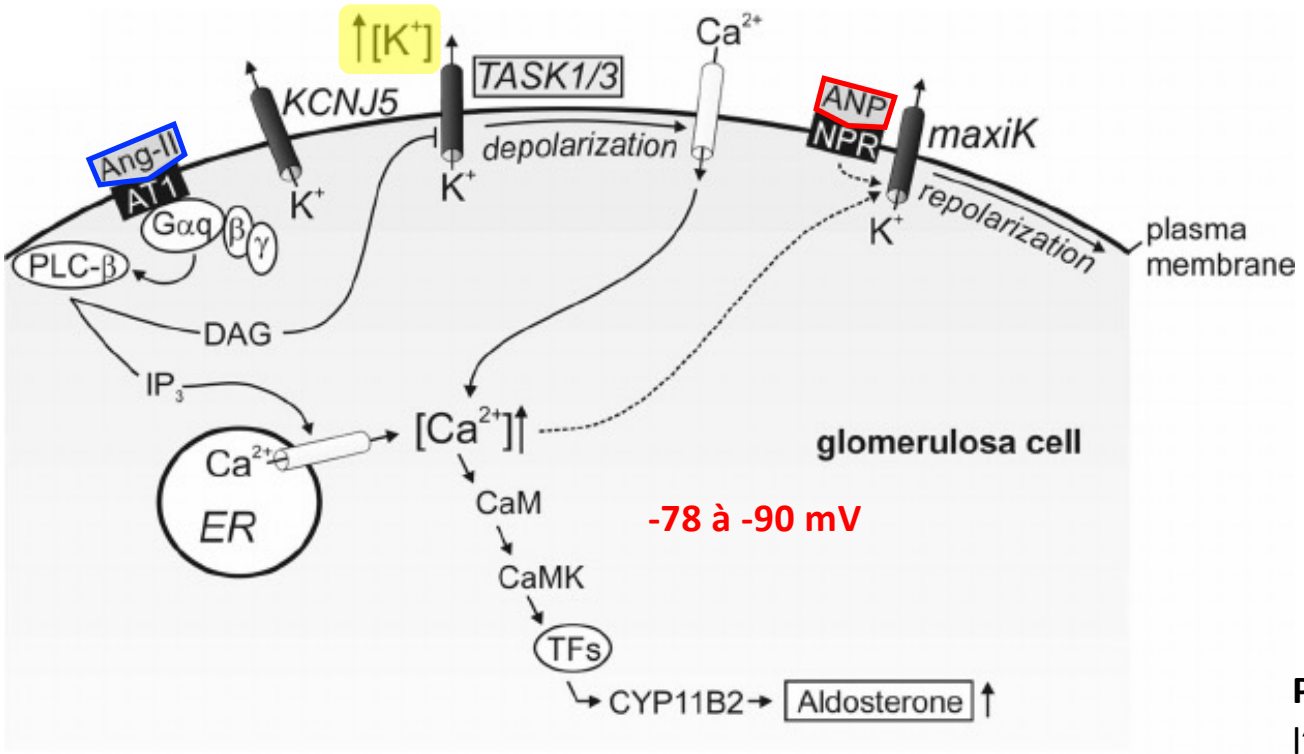
analogue d'aldostérone

Plus spécifique pour le MR (Récepteur Minéralocorticoïde)
--> Bloque moins le AR (Androgène Récepteur)



L'éplérénone possède un prix publique environ 12 fois plus élevé
(Inspra® 25 mg 100 cp à CHF 361.40
vs
Aldactone® 25 mg 100 cp à CHF 29.40).

Facteurs déterminant la sécrétion d'aldostérone par les cellules de la **zone glomérulée**

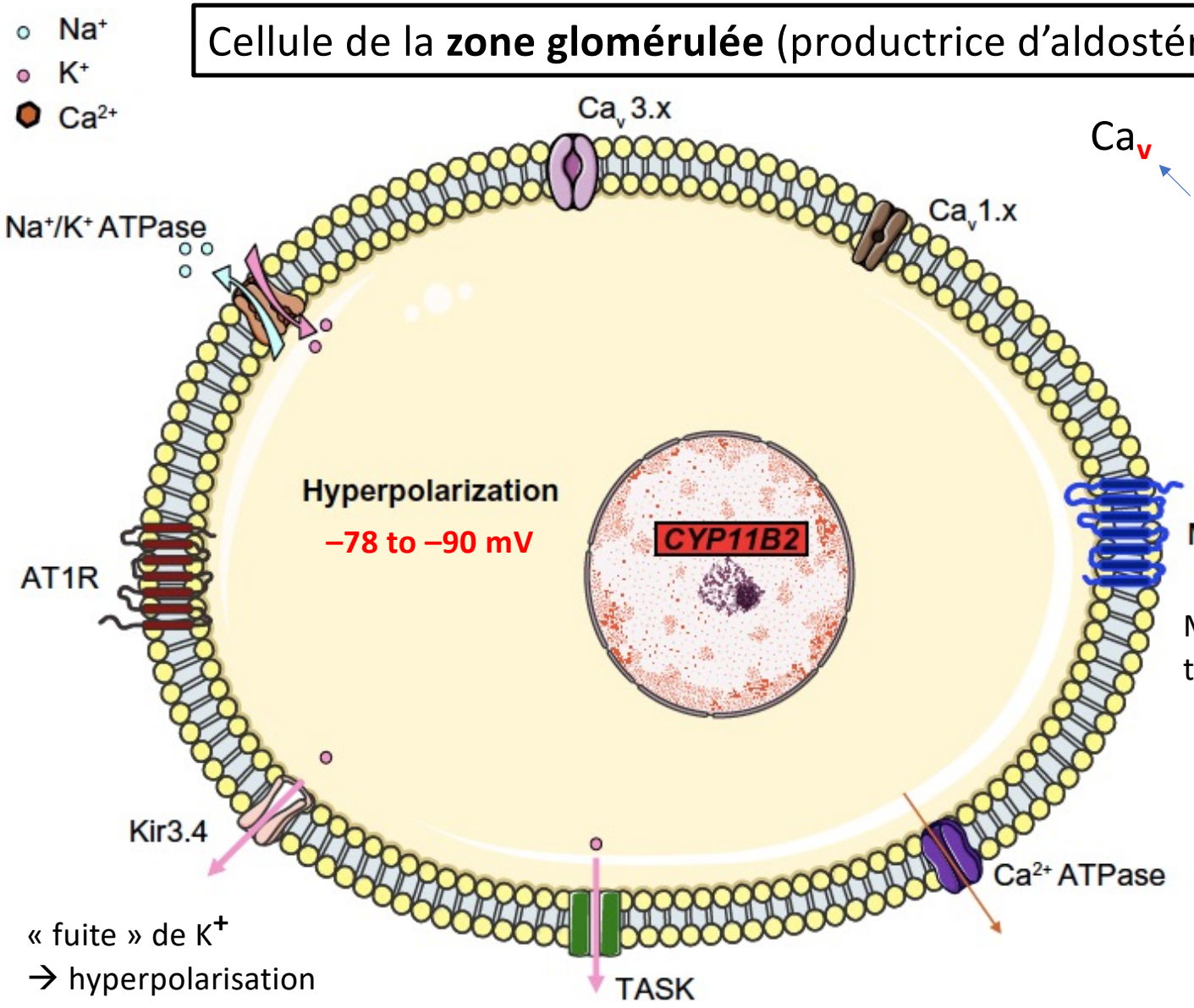


Transcription du gène

- kaliémie
- angiotensine II
- ANP (Atrial Natriuretic Peptide)
- ACTH

Pas de stockage :
l'aldostérone produite est sécrétée

Cellule de la **zone glomérulée** (productrice d'aldostérone)



Ca_v

régulé par le voltage

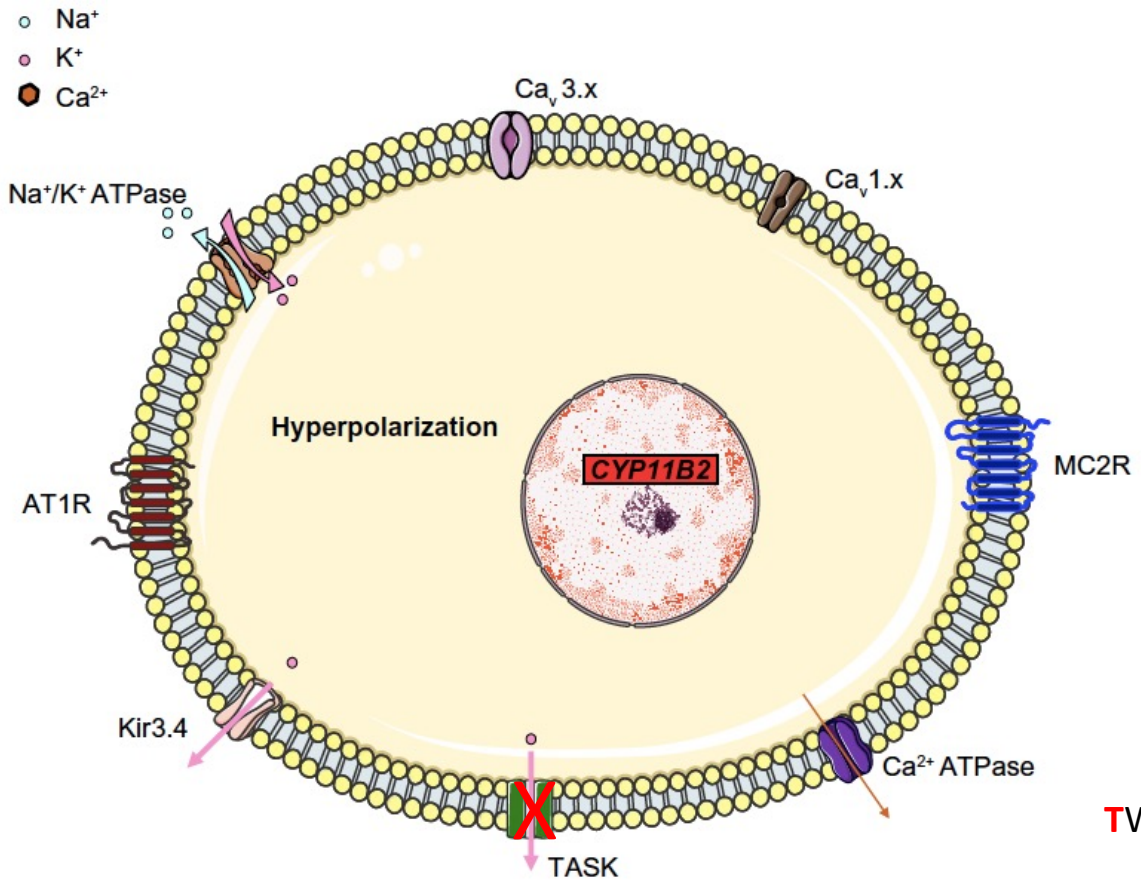
Melanocortin récepteur type 2

« fuite » de K^+

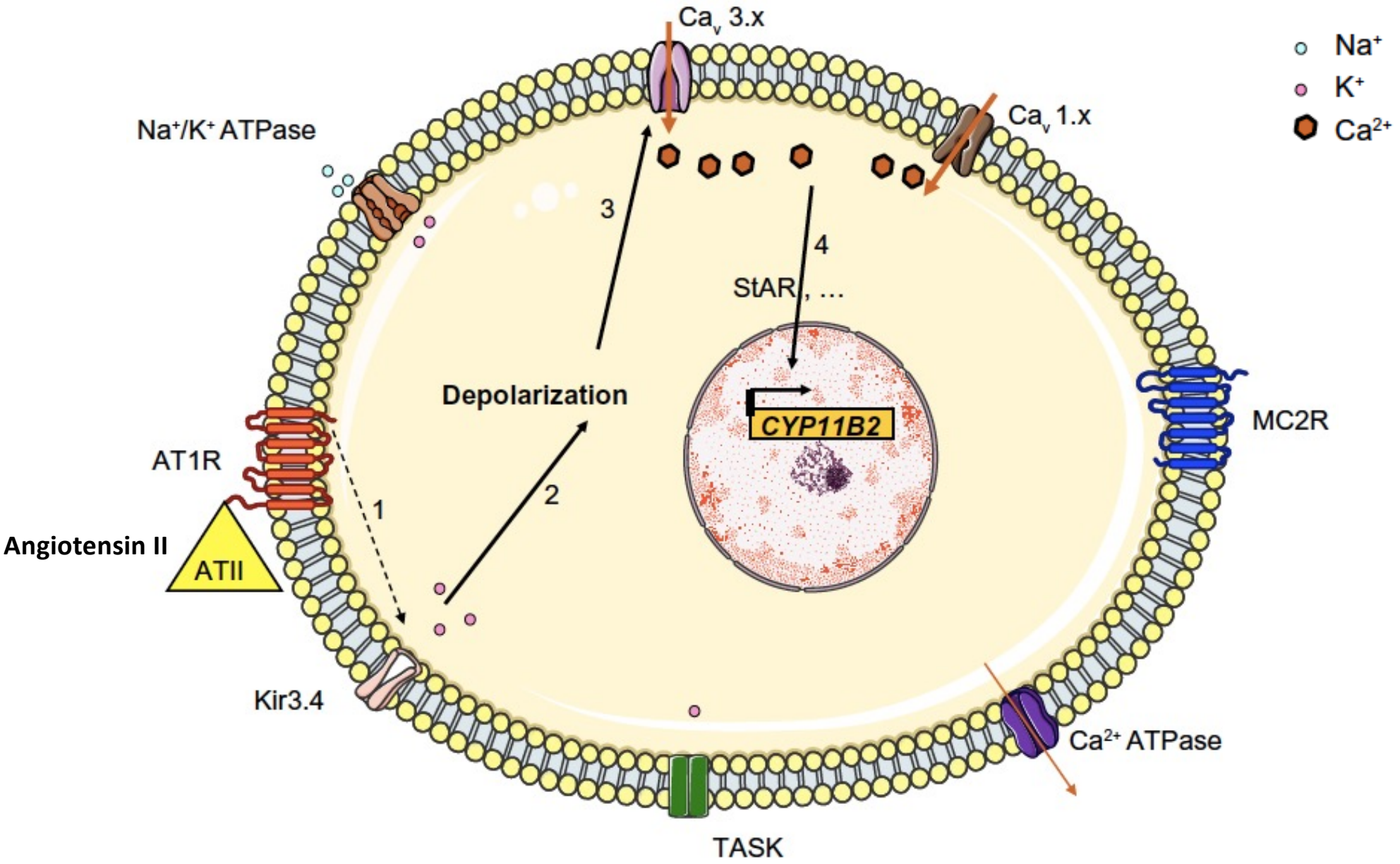
→ hyperpolarisation

TASK channel deletion in mice causes primary hyperaldosteronism

Lucinda A. Davies*, Changlong Hu*, Nick A. Guagliardo†, Neil Sen*, Xiangdong Chen*, Edmund M. Talley*‡, Robert M. Carey†, Douglas A. Bayliss*§¶, and Paula Q. Barrett*¶



TWIK-related acid-sensitive K channel

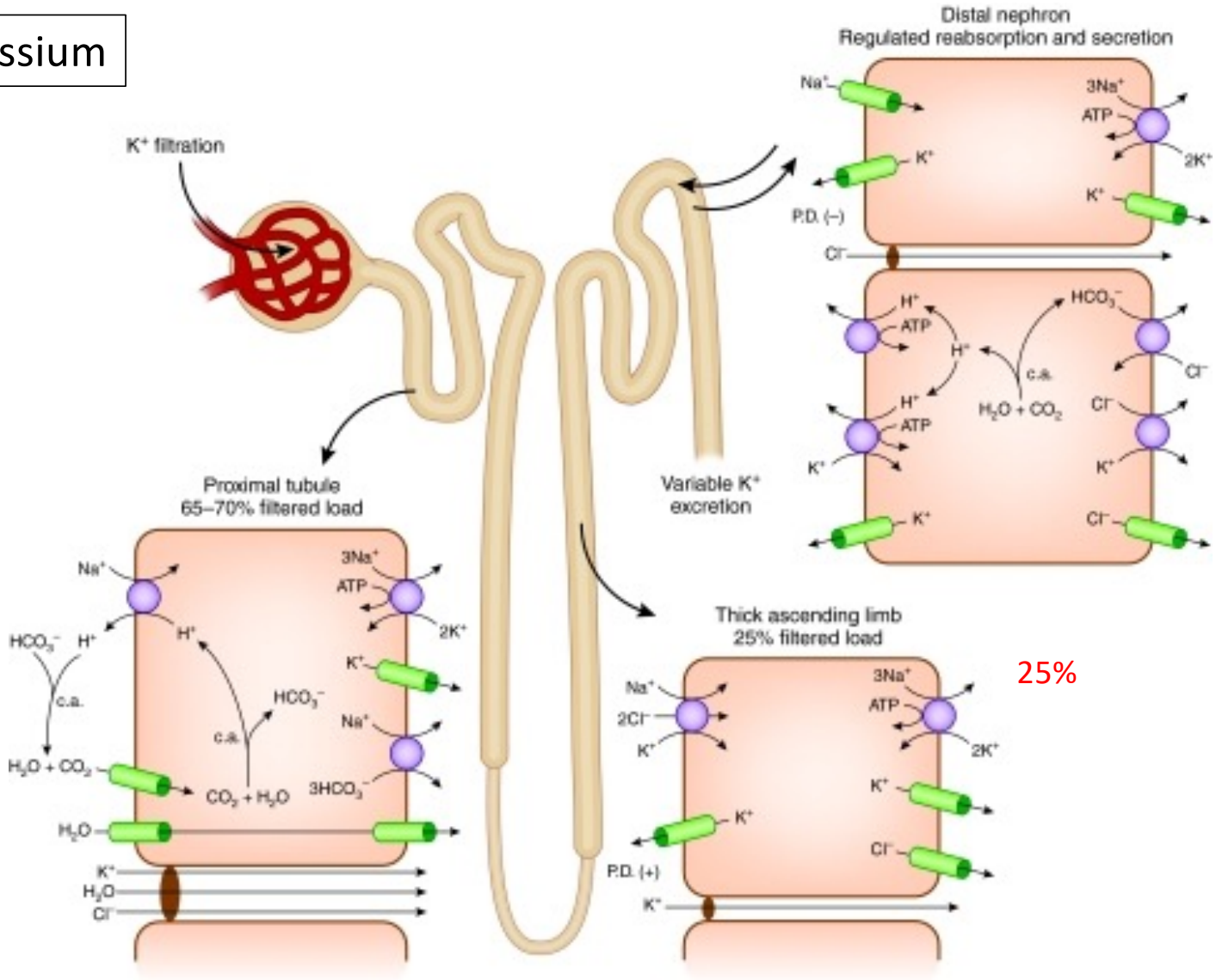

Ca$_v$ 3.x
Na$^+$
K$^+$
Ca^{2+}
Na$^+$/K$^+$ ATPase
Ca$_v$ 1.x
3
4
StAR, …
Depolarization
CYP11B2
MC2R
AT1R
1
2
Angiotensin II
ATII
Kir3.4
Ca^{2+} ATPase
TASK

Comment angiotensine II induit la libération d'aldostérone

On the plasma membrane of the **glomerulosa cell**,
ANG II binds to the **AT receptor** (type 1 ANG II receptor),
which couples through the Gαq –mediated pathway to
phospholipase C (PLC).
Stimulation of PLC leads to the formation of diacylglycerol
(DAG) and inositol 1, 4, 5-triphosphate (IP3).
DAG activates protein kinase C (PKC). IP3 triggers the release
of Ca^{++} from intracellular stores, thus causing a rise in $[Ca^{++}]_i$,
which activates Ca++ -dependent enzymes such as PKC and
Ca++ –calmodulin-dependent protein kinases. These changes
lead to **depolarization of the glomerulosa cell's plasma membrane**,
opening of voltage-activated Ca^{++} channels, and a *sustained* increase
in Ca^{++} influx from the extracellular space. This rise in $[Ca^{++}]_i$ is
primarily responsible for triggering the synthesis (i.e., secretion)
of aldosterone.

Boron

Bilan du potassium



70%

25%

Cellule principale

Cellule intercalaire (type α)